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Caprine Herpesvirus Infection in Switzerland: Some Aspects of its Pathogenicity

By

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With 4 figures and 4 tables

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The existence of caprine herpesvirus (cap HV) outside of California became evident when several kids of a herd in the Bregaglia valley (eastern Switzerland) died of an ulcerative enteritis. Pathological and virological investigations revealed that the causative agent was a caprine herpesvirus (13). No abortions were observed in the infected flock.

Previous investigations on cap HV have been confined to an outbreak of the disease in California (17) and subsequent virological and pathogenetical studies (3,4).

The aim of the present study was to compare the isolated Swiss strain of caprine herpesvirus with that described from California: (I) to confirm its pathogenicity for kids, (II) to determine its abortifacient properties and (III) to find out whether it produces specific lesions in the foetuses.

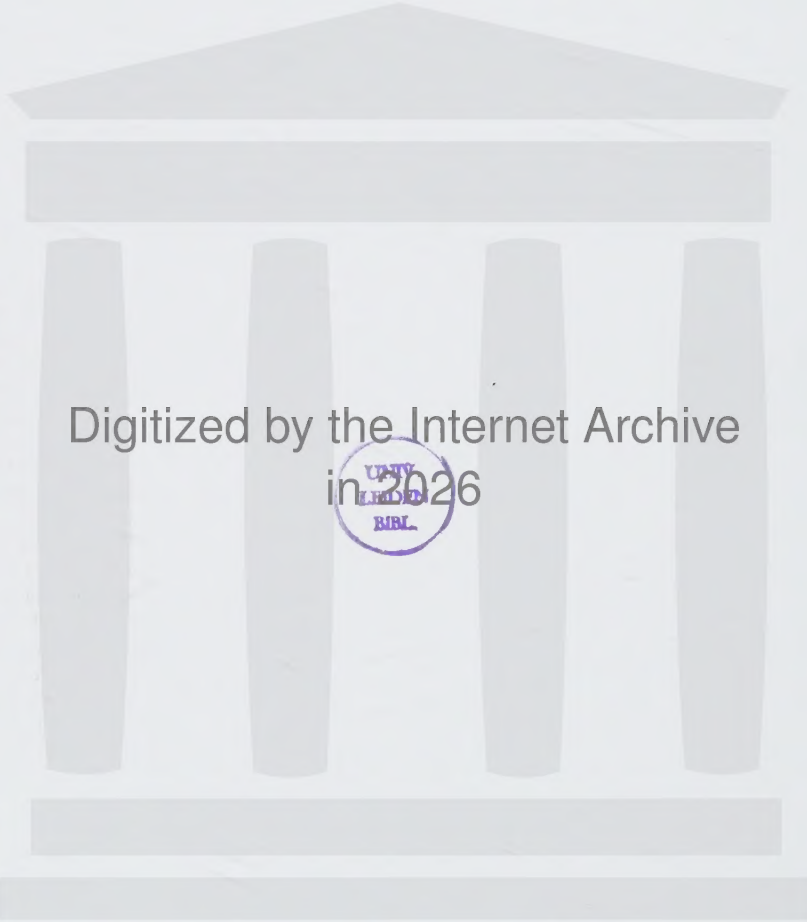
Material and Methods

1. Clinical examination and collection of material for laboratory investigation

1.1. Inoculation of kids

Four two week old male Saanen kids were purchased from a farm a long distance from the area of the reported infection (13). The rectal temperature was recorded twice a day.

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Total red blood cell (RBC) and white blood cell (WBC) count, differential WBC count, haemoglobin and packed cell volume were determined three times a week and compared with the values found by HOLMAN and DEW (10). Serum was taken from all blood samples for determination of neutralizing antibody titres against the virus.

One week after purchase, three of the kids were inoculated intravenously with 2 ml. of a cap HV suspension containing $10^{6.8}$ tissue culture infectious dose 50 % ($TCID_{50}$)/ml. The fourth animal received 2 ml. of supernatant from an uninfected culture of embryonic bovine lung cells, administered intravenously, and the animal was kept separately from the infected kids.

To demonstrate virus shedding, nasal, oral and rectal swabs were collected three times a week on dry, cotton-tipped applicator sticks, which were squeezed out immediately into Eagle's minimal essential medium (MEM) containing 2 % foetal calf serum (GIBCO Bio-Cult, Glasgow, Scotland), 100 IU/ml. penicillin (NOVO Industri A/S Copenhagen, Denmark), 100 µg./ml. streptomycin (NOVO Industri A/S Copenhagen, Denmark) and 150 µg./ml. Gentamycin (Schering Corporation, Kenilworth, N. J. 07033) and then stored at -20°C .

The inoculated kids were killed by injection of a lethal dose of barbiturate on post-inoculation days (p.i.d.) 6, 9 and 26 and were examined post-mortem. For histological examination the relevant tissues were fixed in 4 % buffered formaldehyde. Samples to be used later for virus isolation were stored at -20°C . For demonstration of viral antigen by the indirect fluorescent antibody technique (IFAT), samples of organs were immersed in liquid nitrogen and stored at -70°C .

1.2. Inoculation of pregnant goats

Two goats, purchased on the same farm as the kids, were kept in separate stalls. Gestation was checked by an ultrasonic Doppler instrument (20). Goat 1 was in the third month and goat 2 in the fourth month of pregnancy. Both animals had neutralizing titres of the virus of below 1:2. During the following period the rectal temperatures were recorded twice daily. Temperatures between 38.5°C and 39.7°C are considered as normal according to HÖRNICKE (9). Once a week total RBC and WBC count, differential WBC count, haemoglobin and packed cell volume were determined and compared with the values found by HOLMAN and DEW (10). At the same time serum was collected for determination of serum neutralizing antibody titres to the virus and antibody titers to *Toxoplasma gondii* (Immunofluorescent Test, Hoffmann-La Roche, Diagnostica, Basel, Switzerland).

The goats were inoculated intravenously with 4 ml. of a cap HV suspension containing $10^{6.8}TCID_{50}$ /ml. one week after purchase. Thereafter conjunctival, nasal, oral and vaginal swabs were collected three times a week for demonstration of virus shedding, as mentioned previously. The goats were examined daily by an ultrasonic Doppler instrument for foetal heart sounds to recognize foetal death. Goat 1 died of peritonitis on p.i.d. 17 and goat 2 was killed on p.i.d. 49. The foetuses, placentas as well as tissues from the mother goats were taken for investigation and samples for virus isolation, IFAT and microscopic examination were collected and preserved as mentioned previously. Body cavity fluid from the foetuses was stored at -20°C for demonstration of humoral antibodies to cap HV and liver, kidney, lung, abomasum and placenta were examined bacteriologically.

2. Laboratory investigations

For histological examination the formaldehyde-fixed specimens were embedded in paraffin and sections stained with haematoxylin-eosin. Special staining techniques were applied to sections of foetal lungs including Attwood's phloxine-tartrazine method (2), Martius scarlet blue, and periodic acid Schiff reaction.

The cap HV used for inoculation of the experimental animals, serum neutralization test and IFAT had originally been isolated from Bregaglia and passaged 6 times on embryonic bovine lung cells.

The embryonic bovine lung cell cultures were prepared according to the method described by GOLDSMIT and BARZILAI (8), using Eagle's MEM supplemented with 10 % foetal calf serum (GIBCO, Bio-Cult, Glasgow, Scotland) and 100 IU/ml. penicillin (NOVO Industri A/S Copenhagen, Denmark).

For virus isolation the organs were homogenised with 9 volumes of Eagle's MEM with 2 % foetal calf serum and antibiotics. The suspension was centrifuged at 1,500 g. for 10 minutes and the supernatant was used to inoculate the embryonic bovine lung cell cultures. These were

checked microscopically for an eventual cytopathic effect (CPE) daily until the seventh day after infection.

The level of cap HV neutralizing antibody in sera and foetal body fluids was tested in a microtitre system using 100 TCID₅₀ of cap HV (supernatant of cell cultures) and primary embryonic bovine lung cells. The sera were inactivated at 56 °C for 30 minutes, diluted 1 : 2 to 1 : 5,120 and each dilution distributed in 4 wells. No complement was added. The plates were microscopically checked 24, 48 and 72 hours after infection and the titres calculated according to the method of REED and MUENCH (16).

For electron microscopic examination one virus isolate of a nasal swab from each animal was used for a second passage in embryonic bovine lung cell cultures. When the CPE was advanced, the medium was centrifuged at 10,000 g. for 15 minutes, and the supernatant was centrifuged at 180,000 g. for 45 minutes. The pellet was resuspended in 0.1 M phosphate buffer and negatively stained with 2 % sodium phosphotungstate. Alternatively the monolayers were treated with trypsin (0.125 % w/v) and versene (0.025 %) in phosphate buffered saline (pH 7.3). Cells were spun down at 100 g. for 15 minutes and resuspended in 2.5 % glutaraldehyde in 0.1 M sodium cacodylate, pH 7.4 and centrifuged in microtubes at 500 g. for 20 minutes. This pellet was fixed in 2.5 % glutaraldehyde for 30 minutes, washed in 0.1 M s-collidine buffer, pH 7.4, postfixed in 1 % osmium tetroxide in 0.1 M s-collidine, pH 7.4 for one hour, thereafter dehydrated in ethanol and finally embedded in epon. The sections were stained with uranyl acetate and lead citrate.

The foetal fluid was tested for antibodies by the serum neutralization method and by IFAT. For IFAT, monolayer cultures of embryonic bovine lung cells were infected with cap HV. As a positive control serum from goat 1 with a neutralizing titre of 1 : 1,622 and serum from goat 2 with a titre of 1 : 6,457, were used. Sera obtained from the same animals before inoculation served as negative controls.

IFAT was also used for the demonstration of viral antigen. Cryostat sections of foetal tissue were mounted on slides and either air-dried or fixed with acetone. The positive and negative sera used as control for the demonstration of humoral antibodies were diluted with phosphate buffer 1 : 10 to 1 : 1,000 and placed on the sections. The slides were incubated for 60 minutes at 37 °C and washed three times in phosphate buffer for 5 minutes. Fluorescein isothiocyanate rabbit anti-goat (Nordic immunological products, Tilburg, Netherlands) was applied to the sections and incubated for 60 minutes. The preparations were washed again three times for 5 minutes each in phosphate buffer and counterstained with Evans-Blue. Coverslips were mounted using a solution of phosphate buffer and glycerine (1 : 9) and the sections examined under an ultraviolet light microscope. The adrenal glands of kid II provided a positive control.

Results

1. Inoculation of kids

Kid III contracted severe pneumonia six days before inoculation and was treated with high doses of penicillin for the following four days. It recovered promptly but a cough persisted during the whole experiment. Before inoculation the rectal temperature of kids I and II varied from 39.5 °C to 40.2 °C as did that of kid III, once the clinical signs of pneumonia had disappeared.

After inoculation the rectal temperature rose in kid II to 41.3 °C on p. i. d. 1 and varied henceforth from 40.8 °C to 41.3 °C until the animal was killed on p. i. d. 6. On p. i. d. 4 vesicles became visible on the skin and feet. One day later the vesicles in the skin burst and turned to ulcers (Fig. 1) and the animal seemed to suffer from great pain in its feet. It preferred to lie down and when forced to stand took the same posture as a horse suffering from laminitis.

In kid I the course of the disease was very similar. The rectal temperature rose on p. i. d. 1 to 41.5 °C and varied from 39.6 to 41.7 °C for the following 5 days. Lesions in the skin and feet, as described for kid II, became visible on p. i. d. 4, but no signs of pain in the feet were observed. On p. i. d. 7 the temperature dropped to the preinoculation level and did not rise again before the animal was killed on p. i. d. 9.



Fig. 1. Vesicles and crusts on the nose of kid II on p. i. d. 6

In kid III the rectal temperature began to rise gradually on p. i. d. 1 and reached the maximum of 41.2°C on p. i. d. 4. Thereafter it dropped gradually and from p. i. d. 6 onwards preinoculation values were measured until the animal was killed on p. i. d. 26. Dermal lesions as described for kid II were first observed on p. i. d. 5. In the following period the ulcers healed and were no longer visible after p. i. d. 20.

All three inoculated animals remained alert during the whole experiment and no loss of appetite and no increase in respiration rate was observed. The haematologic values did not change significantly in any kid. The mock infected animal showed no clinical signs of illness (Table 1).

Table 1

Appearance of predominant clinical features and duration of virus shedding of three inoculated kids (numbers refer to p. i. d.)

	fever	lesions of skin and feet first observed	necropsy	viral recovery from swabs		
				nasal	oral	rectal
kid I	1-7	4	9	5; 7	5; 9	7
kid II	1-6	4	6	2; 5	5	5
kid III	1-6	5	26	5; 7	7	5; 7
kid IV (mock infected)	no fever	no lesions	not done	no virus isolated		

The postmortem examination of kid II, which was killed on p. i. d. 6, revealed ulcers in the mucous membranes of mouth, oesophagus, rumen, caecum, colon and urinary bladder as well as the lesions in the skin and feet described above. Histological examination of the skin showed some deep focal necrosis, infiltrated by neutrophils and macrophages. Haemorrhaging had

occurred at the base of the necrosis and the adjacent tissue was locally infiltrated by lymphocytes, neutrophils and monocytes. Ballooning degeneration and a few eosinophilic intranuclear inclusion bodies were found in epithelial cells delimiting the necrosis. The ulcers found in the mucous membranes were similar to those in the skin.

On the feet, the vesicles showed a low degree of inflammatory reaction. As in the skin, ballooning degeneration of epithelial cells and intranuclear inclusions were observed in the stratum germinativum near to the lesion (Fig. 2).

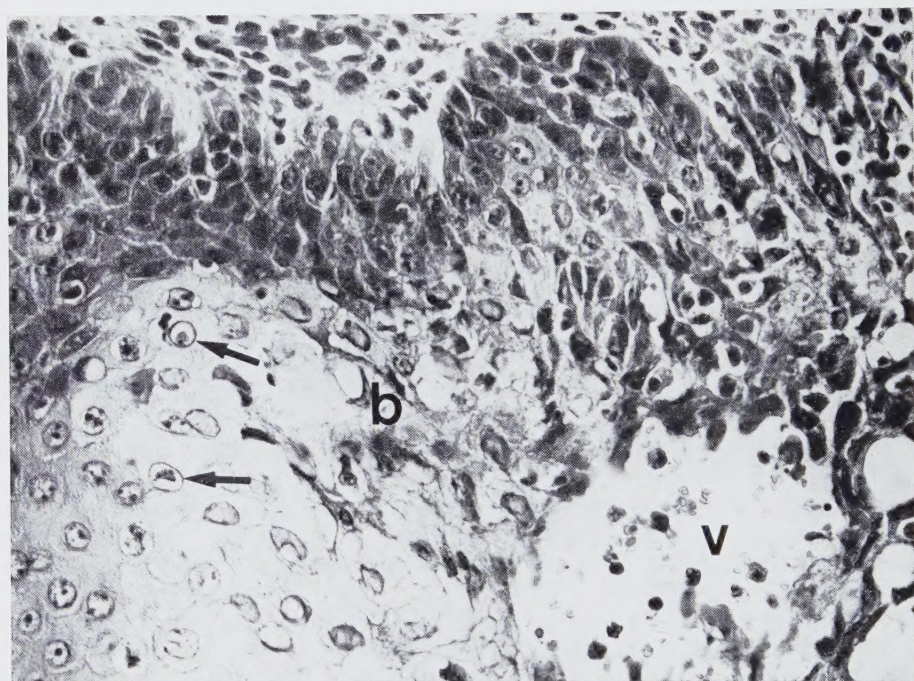


Fig. 2. Dermatitis in the coronary groove: Ballooning degeneration (b), intranuclear inclusions (arrows), vesicle formation (v) on p. i. d. 6 H. E. $\times 420$

In the adrenal cortex necrotic foci with central haemorrhages and slight infiltration of neutrophils were found. In the periphery of these lesions eosinophilic intranuclear inclusions were present (Fig. 3). Furthermore, we observed a mild chronic non-purulent interstitial pneumonia, petechiae in the endocardium and skeletal muscles and a mild non-purulent conjunctivitis.

In kid I, killed on p. i. d. 9, ulcers were found in skin, feet and jejunum. In general the histological feature of these alterations was similar to the findings in kid II, but infiltration of monocytes and neutrophils was more distinct and in the adjacent tissue fibroblasts began to proliferate. However, inclusion bodies were not found in any organ examined from this animal. Petechiae were present in the skeletal muscles and urinary bladder, and a mild non-purulent rhinitis and an acute focal purulent bronchopneumonia of the right apical lobe were observed.

Autopsy of kid III, killed on p. i. d. 26, revealed a chronic purulent bronchopneumonia in the apical and middle lobes, probably occurring as a consequence of the severe pneumonia 6 days before inoculation. Histological examination revealed a mild chronic non-purulent rhinitis, a mild focal non-

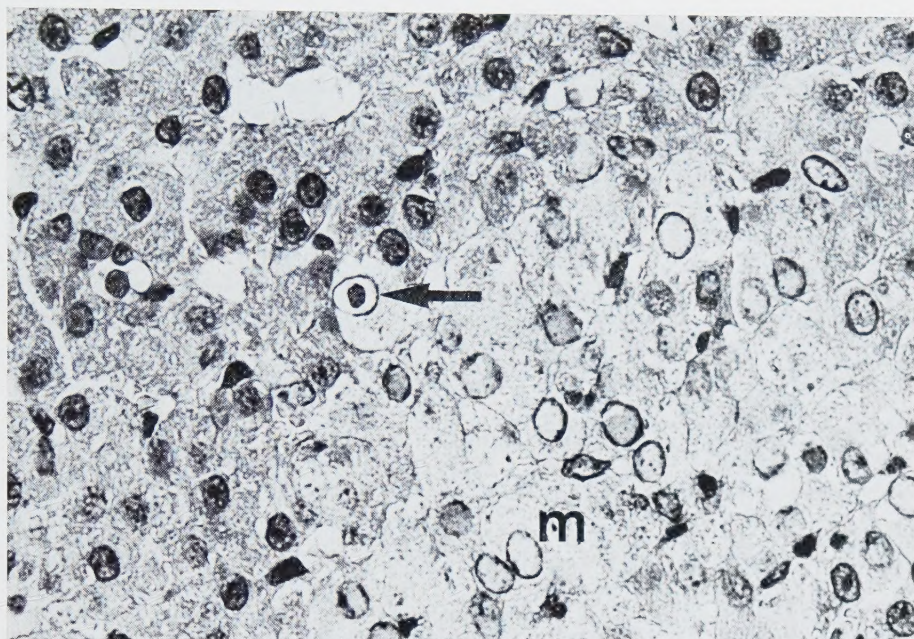


Fig. 3. Adrenal cortex: Transition from normal (left side) into degenerating tissue (right side). Margination of chromatin (m) and intranuclear inclusion body (arrow) on p. i. d. 6 H. E. $\times 680$

purulent conjunctivitis, a chronic focal dermatitis with eosinophilic infiltration and some foci of calcification in the adrenal cortex.

The results of the virological examination of swab material are shown in Table 1. We succeeded in recovering virus between p. i. d. 2 and 9. Virus shedding could be demonstrated for all the locations examined. Nasal virus-isolates from all 3 animals were identified as herpesviruses by electron-microscopy (Fig. 4).

From kid II we succeeded in isolating the virus from pooled muscle and spleen as well as from pooled liver, kidney and intestines. From kid I the virus could only be isolated from the skin.

In kid II, which was killed first, no immunological response was observed, but in the remaining two animals the titre began to increase one week after inoculation and reached its peak in kid III on p. i. d. 19 (Table 2).

2. Inoculation of pregnant goats

2.1. Findings in the goats

In both animals the rectal temperature was elevated above the normal from p. i. d. 2 to 5. Diarrhoea was observed in goat 1 from p. i. d. 11 to 13 and in goat 2 from p. i. d. 6 to 9.

Haematological examination revealed a relative lymphopenia on p. i. d. 7 with 21 % lymphocytes for goat 1 and 25 % for goat 2. The total WBC count yielded no significant changes. The other haematological values remained at normal levels. On p. i. d. 4 no foetal heart sounds could be detected in goat 1. A day later the foetuses (twins) were removed by caesarean section to prevent autolysis. Despite intensive treatment over four days with high doses of penicillin the animal died of postoperative peritonitis on p. i. d. 17. At necropsy we found some sharply delimited ulcers with a red base in the oral cavity

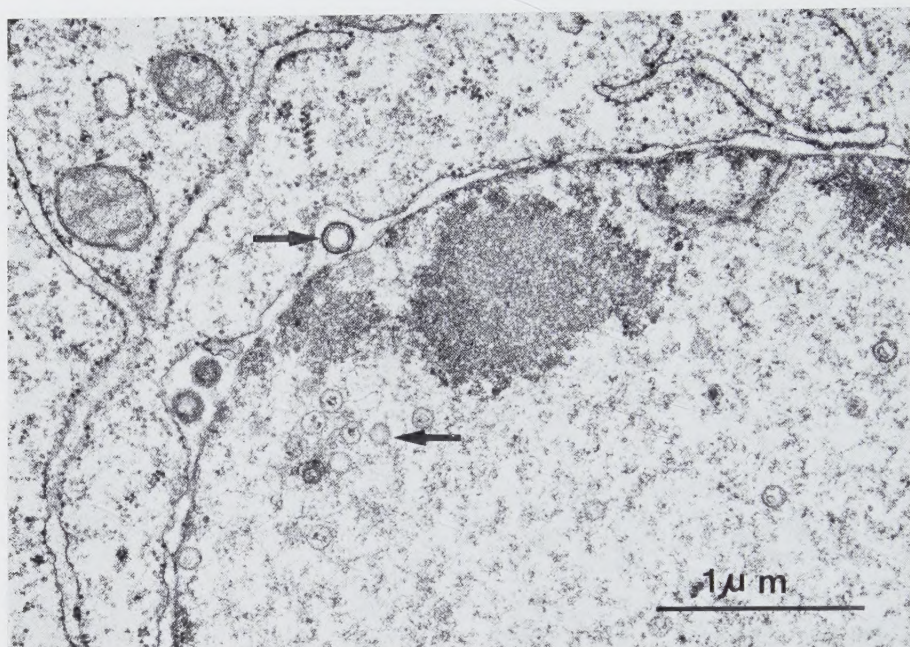


Fig. 4. Bovine embryonic lung cells in culture infected with a nasal virus isolate: Naked virions (arrow) in the nucleus and enveloped viruses (arrow) in the perinuclear space $\times 27,000$

Table 2

Immunological response of three inoculated kids (neutralizing antibody titres)

p. i. d.	0	5	7	9	12	14	16	19	21	23	26
kid I	< 2 ¹	< 2	13	224							
kid II	< 2	< 2									
kid III	< 2	< 2	69	224	813	1023	1280	4074	1280	1023	813
kid IV (mock infected)	< 2	< 2	< 2	< 2	< 2	< 2	< 2	< 2	< 2	< 2	< 2

¹ = reciprocal value of neutralizing antibody titer

and the rumen, and ulcers covered with yellow fibrin in the caecum and colon ascendens. Numerous small, sharply delimited, yellow patches of thickened epithelium were present in the oesophagus. Microscopic examination revealed focal necrosis in the adrenal cortex and some small foci of ballooning degeneration in the epithelium of the oesophagus. All lesions were accompanied by slight infiltrations of lymphocytes, neutrophils, macrophages and marked fibroplasia. The infiltration was more pronounced in the necrotic foci of the caecum and colon ascendens. No inclusion bodies were found in the tissues examined.

Goat 2 aborted triplets on p. i. d. 7. One day before abortion normal foetal heart sounds were recorded. The placentas were partly retained. To prevent a bacterial infection, tetracycline was administered intrauterinely. No complication was observed during the puerperium.

At necropsy of goat 2 on p. i. d. 49, we found nodules originating from parasites in the lung and a slight degree of eosinophilic infiltration in the abomasum and the small intestines. In the adrenal cortex focal fibrosis with

formation of lymph follicles was observed. Virus shedding could be demonstrated in conjunctival, nasal, oral and vaginal swab material from p. i. d. 3 to 10 in goat 1 and from p. i. d. 6 to 13 in goat 2 (Table 3). Nasal virus isolates from both goats were identified as herpesviruses by electronmicroscopy.

We did not succeed in isolating virus from inner organs of either goat.

The neutralizing antibody titre began to rise in both animals on p. i. d. 7 and reached its highest level of 1 : 6,457 in goat 2 on p. i. d. 43 (Table 4).

The toxoplasma antibody titre did not change significantly in either of the goats.

Table 3

Virus isolations from swabs of the two inoculated goats

	specimen	p. i. d.				
goat 1	conjunctival	3	6	8		
	nasal	3	6	8	10	
	oral		6	8	10	
	vaginal	3	6	8		
goat 2	conjunctival		6	8	10	
	nasal			8		
	oral		6			
	vaginal		6	8	10	13

Table 4

Immunological response in pregnant goats after inoculation

post-inoculation day	neutralizing antibody titers in sera	
	goat 1	goat 2
0	1 : 2	1 : 2
7	1 : 93	1 : 13
13	1 : 1622	1 : 4074
20		1 : 3631
29		1 : 5120
36		1 : 5120
43		1 : 6457

Correction: In the 2nd and 3rd column, 1st line, must read > 1 : 2.

2.2. Findings in the foetuses

The two foetuses of goat 1 were oedematous and of a brown colour. The body cavities contained a large amount of watery, dark brown fluid. Neither macroscopic nor microscopic lesions were found. The histological examination of the placenta yielded only moderate autolytic changes. There was no indication of an aspiration of amniotic fluid (1).

We succeeded in isolating the virus from the placenta, but not from inner organs of the foetuses. Bacterial or mycotic infection was excluded.

Two foetuses from the triplets of goat 2 were moderately autolytic. Their body cavities contained a large amount of dark-red watery fluid. The lung of the first foetus was irregularly dark-red discoloured and in the lung of the second foetus small red foci were found. The third foetus showed no signs of autolysis and no gross lesions were visible. Microscopic lesions were confined to the lungs of the three foetuses. They consisted of foci of dilated blood vessels, often associated with haemorrhage. There was no indication of an aspiration of amniotic fluid (1).

Virus was recovered from the placenta and from the lung of the first foetus. Attempts to isolate pathogenic bacteria or fungi yielded negative results.

No humoral antibodies could be demonstrated in the foetuses of either goat, either by serum neutralization test or by IFAT. Furthermore, the use of IFAT to demonstrate viral antigen in inner organs of the foetus yielded negative results. In the placenta non-specific fluorescence, which could not be eliminated by counterstaining with Evans blue, made an evaluation of IFAT impossible.

Discussion

Pathogenicity of the cap HV strain Bregaglia for kids was confirmed by our experiments. The clinical features of the disease and post-mortem findings were compatible with the observations in the spontaneous outbreak (13). The demonstration of extensive virus shedding, the presence of ulcers in skin and mucous membranes of the alimentary tract, focal necrosis of the adrenal cortex, the recovery of viruses from damaged organs and the serological response of the animals confirm the viral aetiology of the disease.

The lesions in oral cavity, oesophagus, rumen, caecum, colon ascendens and adrenals in goat 1 were comparable to the findings in the kids and therefore may also be attributed to the caprine herpesvirus.

In kid III and goat 2 which were killed and examined on p. i. d. 26 and 49, respectively, the only lesions which might be associated with cap HV infection were found in the adrenals, consisting of foci of calcification in the kid and focal fibrosis with formation of lymph follicles in the adult goat. Either the virus did not produce further lesions in the organs examined or the injured tissues were regenerated completely as in the case of the dermal ulcers in kid III.

Our main objective was to show that the herpesvirus we have isolated (13) may also cause abortion in the goat as was reported for the "Californian" virus. In fact foetal death or abortion occurred in both the inoculated goats. It seems unlikely that this was due to some infective agent other than the virus since neither bacteria nor fungi were isolated from the foetuses or placentas, nor was a significant change in the antibody titre against *Toxoplasma gondii* registered for the dams. Statistically we can say with 99 % confidence that the event of two abortions in two goats is not compatible with a frequency of abortion less than 7.07 %. No reliable data are available for a mean frequency of abortion in goats. LEECH and SELLERS found that the mean percentage of abortion in the Yorkshire sheep does not exceed 4.28 % (12) and on the farm from which the experimental animals originated 3 abortions were recorded out of 364 deliveries on term. From this we conclude that the goats aborted due to the intravenous inoculation of the cap HV.

Virus could be recovered from the placentas of both dams and from the lung of one foetus, suggesting that the herpesvirus may cross the placental barrier and gain access to the foetus. However, viral antigen could not be localised by IFAT in the inner organs of the foetuses. Foetal lesions, as described for herpesvirus infections in cattle (11), swine (21) or horses (6, 15) were not observed in the caprine foetuses. We suggest, therefore, that foetal death was caused by the disease of the mother and not by direct viral injury to the foetus. This assumption is supported by the facts that febrile reaction and foetal death coincided in goat 1 and that the interval between both events was very short in goat 2. However, the absence of viral lesions in the foetuses examined here does not completely exclude the possibility that in other cases cap HV could induce abortion by damage to the foetus.

In cattle for example, abortion due to bovine herpesvirus 1 (infectious bovine rhinotracheitis) is attributed to foetal infection, which is normally manifested as focal necrosis of the inner organs of the foetus (14), and there is

usually a lag phase observed between infection of the mother and abortion (7). However, cases of abortion due to infectious bovine rhinotracheitis have been reported, where foetal death coincided with the dam's illness and no visible injury in the foetus was found (18), a situation analogous to that observed here with cap HV.

Virus shedding, an important factor in propagation of infection, was extensive in both adult goats and kids towards the end of the febrile phase of the disease. However preliminary serological examinations have shown that the infection does not seem to be widespread in the Bregaglia region and so far we have no indication of the presence of the disease in other parts of Switzerland (19).

A tentative diagnosis of a cap HV infection may be based on the observation of ulcers in the skin and oral cavity of kids or goats. Pathological examination of such animals may provide further indications of this disease such as the demonstration of ulcers in the mucous membranes of the alimentary tract and urinary bladder as well as necrotic foci in the adrenals. However the aetiology of this disease can not be confirmed unless the cap HV can be reisolated from damaged organs or intranuclear inclusions of the Cowdry type A are demonstrated within the lesions. Such intranuclear inclusions were rare in our experiments and were observed only in the kid which had been killed first. This suggests that a similar situation exists for cap HV as for the bovine herpesvirus 1 and that intranuclear inclusions are present only for a short time (5). The fact that cap HV infections can only be diagnosed with certainty by means of histological and virological methods may explain the rareness of reports on cap HV infections.

If we compare our findings with the results reported by BERRIOS et al. for the experimental investigations with the "Californian" cap HV isolate (3), we note very similar features in the results of the virological examinations, but differences in the clinical manifestation of the disease. BERRIOS observed the disease to be fatal in intravenously inoculated kids, in contrast to our findings, where none of the kids became critically ill. Furthermore, in his experiments the pregnant goats, which had been inoculated with "Californian" cap HV, aborted several weeks after the end of the febrile response, whereas we found a close temporal relationship between the dam's disease and foetal death or abortion. It is however difficult to interpret the discrepancies in the clinical aspects of the disease, since these may arise from differences in the experimental conditions such as the environment and the age and breed of the goats.

In the above experiments we have shown that the Swiss strain of cap HV is pathogenic for young animals and induces abortion when used to inoculate pregnant goats. In these features it shows parallels with "Californian" cap HV and it seems probable that the two strains are closely related.

Summary

Experimental investigations on a caprine herpesvirus (cap HV) isolate from the Bregaglia valley (Switzerland) are described and the results compared with reports on a Californian cap HV. Pathogenicity of the cap HV strain Bregaglia for kids was confirmed by inoculation with a virus preparation. Abortifacient properties of the virus were demonstrated by intravenous inoculation of the agent into two pregnant goats. After a febrile reaction the goats aborted. Virus was recovered from the placentas and the lung of one foetus, but no specific lesions could be demonstrated in the foetuses and an attempt to demonstrate viral antigen in foetal organs yielded negative results.

Acknowledgements

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Zusammenfassung

Caprine Herpesvirusinfektion in der Schweiz:

Einige Aspekte ihrer Pathogenität

Ein caprines Herpesvirus (cap HV) Isolat aus dem Bergell (Schweiz) wurde experimentell untersucht und die Ergebnisse mit den Berichten über ein kalifornisches cap HV verglichen.

Die Pathogenität des cap HV Stammes Bregaglia für junge Ziegen wurde mittels Inokulation einer Virusaufbereitung bestätigt.

Die abortauslösende Wirkung des Virus wurde durch intravenöse Applikation des Agens in zwei graviden Ziegen nachgewiesen. Die Ziegen abortierten nach einer fieberhaften Reaktion. Das Virus konnte aus den Plazenten und der Lunge eines Fötus isoliert werden, ohne daß spezifische Läsionen bei den Föten nachgewiesen wurden. Der Versuch virales Antigen in fötalen Organen nachzuweisen erbrachte negative Ergebnisse.

Résumé

Infection chez la chèvre avec un virus Herpes en Suisse:

Quelques aspects de sa pathogénicité

Un virus Herpes caprin (cap HV) isolé dans le val Bregaglia (Suisse) a été examiné expérimentalement et on a comparé les résultats avec ceux d'un virus californien (cap HV).

La pathogénicité de la souche cap HV Bregaglia pour des cabris a été confirmée avec une inoculation d'une préparation virale.

L'action abortive du virus a été mise en évidence par une application intraveineuse du virus chez deux chèvres en gestation. Les chèvres ont avorté après un épisode de fièvre. Le virus a pu être isolé à partir du placenta et des poumons d'un fœtus sans lésions spécifiques constatées. La recherche d'un antigène viral dans les organes du fœtus est demeurée négative.

Resumen

La infección por el virus herpes caprino en Suiza:

Algunos aspectos de su patogenia

Se examinó experimentalmente un virus herpes caprino (VH cap), aislado en el valle de Bregaglia (Suiza), comparándose los resultados con los de un VH cap californiano.

Se confirmó la patogeneidad del VH cap estirpe Bregaglia para cabritos mediante la inoculación de una preparación virósica.

La acción abortiva del virus se probó por medio de la aplicación intravenosa del agente en dos cabras grávidas. Las cabras abortaron tras una reacción febril. El virus se pudo aislar de las placentas y del pulmón de un feto, sin que se pudieran identificar lesiones específicas en los fetos. Resultados negativos trajo consigo el intento de identificar antígeno viral en los órganos fetales.

References

1. ANDREWS, E. J., 1974: Pulmonary pathology in stillborn non-human primates. *J. Am. Vet. Med. Assoc.* 164, 715—718.
2. ATTWOOD, H. D., 1958: The histological diagnosis of amniotic fluid embolism. *J. Path. Bact.* 76, 211—215.

3. BERRIOS, P. E., D. G. MCKERCHER and H. D. KNIGHT, 1975: Pathogenicity of a caprine herpesvirus. *Am. J. Vet. Res.* 36, 1763—1769.
4. BERRIOS, P. E., and D. G. MCKERCHER, 1975: Characterization of a caprine herpesvirus. *Am. J. Vet. Res.* 36, 1755—1762.
5. CRANDELL, R. A., W. J. CHEATHAM, and F. D. MAURER, 1959: Infectious bovine rhinotracheitis — The occurrence of intranuclear inclusion bodies in experimentally infected animals. *Am. J. Vet. Res.* 20, 505—509.
6. GEISEL, O., E. BOCH and P. A. BACHMANN, 1977: Zum Vorkommen und zur Histogenese unterschiedlicher fetaler Pneumopathien beim Virusabort des Pferdes. *Berl. Münch. Tierärztl. Wochenschr.* 90, 429—432.
7. GIBBS, E. P. J., and M. M. RWEYEMAMA, 1977: Bovine herpesvirus. Part I. Bovine herpesvirus 1. *Vet. Bull.* 47, 317—343.
8. GOLDSMIT, L., and E. BARZILAI, 1975: Propagation of bovine viral diarrhea viruses in bovine fetal lung cultures. *Am. J. Vet. Res.* 36, 407—412.
9. HOERNICKE, H., 1976: Thermophysiology. In: *Lehrbuch der Veterinärphysiologie*, 370—371, Hrsg. SCHEUNERT A., und A. TRAUTMANN, Verlag Paul Parey, Berlin und Hamburg.
10. HOLMAN, H. H., and S. M. DEW, 1963: The blood picture of the goat. *Res. Vet. Sci.* 121—130; idem 1964, 5, 274—285; idem 1965, 6, 245—253; idem 1965, 6, 510—521.
11. KENNEDY, P. C., and W. P. C. RICHARDS, 1964: The pathology of abortion caused by the virus of infectious bovine rhinotracheitis. *Pathol. Vet.* 1, 7—17.
12. LEECH, F. B., and K. C. SELLERS, 1959: A second survey of losses associated with pregnancy and parturition in Yorkshire sheep. *J. agric. Sci.* 52, 117—124.
13. METTLER, F., M. ENGELS, P. WILD and A. BIVETTI, 1979: Herpesvirusinfektion bei Zicklein in der Schweiz. *Schweiz. Arch. Tierheilkd.* 121, 655—662.
14. OWEN, N. V., T. L. CHOW, and J. A. MOLELLO, 1968: Infectious bovine rhinotracheitis: Correlation of fetal and placental lesions with viral isolations. *Am. J. Vet. Res.* 29, 1959—1965.
15. PRICKETT, M. E., 1970: The pathology of disease caused by equine herpesvirus 1. *Proc. 2nd int. Conf. equine infectious diseases*, Paris 1969, 24—33.
16. REED, L. J., and H. MUENCH, 1938: A simple method of estimating fifty percent endpoints. *Am. J. Hyg.* 27, 493—497.
17. SAITO, J. K., D. H. GRIBBLE, D. E. BERRIOS, H. D. KNIGHT, and D. G. MCKERCHER, 1974: A new herpesvirus isolate from goats: preliminary report. *Am. J. Vet. Res.* 35, 847—848.
18. STUBBINGS, D. P., and I. R. D. CAMERON, 1981: Bovine abortion associated with infectious bovine rhinotracheitis virus infection. *Vet. Rec.* 108, 101—102.
19. WALDVOGEL, A.: Vorläufige seroepizootologische Untersuchungen über die Verbreitung der caprinen Herpesviren in der Schweiz. *Schweiz. Arch. Tierheilkd.* (in press)
20. WEISS, G., 1975: Möglichkeiten und Grenzen der Graviditätsdiagnose bei Haustieren mit Hilfe der Ultraschall-Doppler-Technik. *Schweiz. Arch. Tierheilkd.* 117, 123—134.
21. WOHLGEMUTH, K., P. F. LESLIE, D. E. REED, and D. K. SMIDT, 1978: Pseudorabiesvirus associated with abortion in swine. *J. Am. Vet. Med. Assoc.* 172, 478—479.

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